Foreign Animal Disease Report

United States Department of Agriculture
Animal and Plant Health Inspection Service
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Emergency Programs

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In This Issue

Emergency Field Operations

Current Events

Screwworms in imported dog. On August 7, 1987, Federal Emergency Field Force offices were authorized by Veterinary Services (VS) to coordinate and direct activities designed to eliminate the threat of screwworms (Cochiliomyia hominivorax) in the southeastern United States. This action was considered necessary when a dog being returned to the United States from Honduras by way of New Orleans was found infested with screwworm larvae upon arrival at its home in Panama City. Larvae were submitted to the National Veterinary Services Laboratories in Ames, Iowa. An earlier introduction of screwworms into the United States this year was reported in the previous issue of Foreign Animal Disease Report (15-3:1).

On August 10, 1987, offices were opened in New Orleans, Louisiana, and Panama City, Florida. Public Affairs Specialists reported to each office to heighten public awareness of the project by stimulating media coverage by local newspapers and radio and television stations. Press kits with press releases, fact sheets, leaflets, and posters were distributed to the media. Videotapes, black and white prints, and color slides were also provided. Contacts were made, and fly collection kits for submitting specimens were distributed to county extension agents, accredited veterinarians, stockyard operators, feed stores, veterinary clinics, and many livestock owners and producers within a 50-mile radius and along Interstate highway 10. Fish and wildlife officials were contacted in Alabama, Florida, Louisiana, and Mississippi. They were advised of the situation and of the need for their assistance in surveillance activities. Specimen collection kits were also provided to fish and wildlife field personnel. Letters notifying accredited veterinarians of the threat of screwworms and requesting their assistance in submitting and reporting suspicious cases were mailed to all veterinarians in Alabama, Florida, Louisiana, and Mississippi by the VS area offices. The intensive surveillance and press

coverage continued until August 21, 1987. At that time, the number of people involved in these activities was reduced to those required to receive reports, investigate reported cases, and identify specimens. On September 5, 1987, the office in Panama City was closed and subsequent surveillance activities were conducted through county agents, accredited veterinarians, and local, State, and Federal employees in the area.

On August 15, 1987, sterile screwworm flies were first released over a 50-mile radius of New Orleans, Panama City, and Interstate highway 10 at the rate of 8 million sterile flies per week. This activity was conducted from the New Orleans airport. Sterile screwworm pupae were received by commercial air carrier from Mexico biweekly and were boxed at New Orleans and dispersed by contract aircraft. This activity continued through the week of September 24, 1987, to complete 6 weeks of sterile fly release, as recommended by entomologists. The office at New Orleans, Louisiana, was closed on September 25, 1987. Intensified surveillance activities have not revealed any evidence of screwworm infestation in the United States.

Avian influenza and exotic Newcastle disease alert. A press release alerting poultry owners and the public of the threat of avian influenza was being prepared at the time this issue of FADR was being written. Similar public awareness activities including radio coverage were also being prepared to alert the public of the threat of exotic Newcastle disease, especially through the introduction of exotic pet birds. A video tape is being produced to alert poultry producers, market operators, handlers, and others visiting poultry farmers of the methods and requirements of bio-security in preventing avian influenza and other diseases of poultry.

Video tapes on foreign animal diseases. Video tapes have been produced on foreign animal diseases from 16mm sound color films already available from VS. (See 13-3:9.) These tapes can be purchased from WRS Motion Picture and Video Laboratory, 210 Semple Street, Pittsburgh, Pennsylvania 15213 (area 412-687-3700).

Training. During the period September 21-October 6, 1987, 18 veterinarians completed a foreign animal diseases training course conducted by Emergency Field Operations (EFO) at the National Veterinary Services Laboratories in Ames, Iowa: Plum Island, New York: and Hyattsville, Maryland. Also, during the period September 9-11, 1987, 17 veterinarians were trained in wildlife diseases at a seminar held at the University of Georgia, Athens, Georgia.

In September 1987, a test exercise of the Recorded Emergency Animal Disease Information (READI) system was completed, followed by a critque on October 8, 1987, in Hyattsville, Maryland, involing VS regional representatives and VS headquarters staffs. The READI system uses computers to rapidly transmit data for the management of emergency animal disease investigations and disease control and eradication operations. (Dr. M. A. Mixson, 301-436-8073)

Foreign Animal Disease Update

On July 27, 1987, Chile reported that a total of 120 herds were affected with **foot-and-mouth disease (FMD)**, virus type O_1, during February 1987. Approximately 30,000 animals were slaughtered by the Government in an effort to stamp out the disease. Owners of the animals were indemnified at a legal rate calculated from the average market price of the animals minus the costs attributable to the eradication program. Chilean authorities continue to closely monitor animals in the affected areas for signs of the disease.

Italy's current FMD epidemic, which began in November 1984, seems to be slowing down. Types O, A, and C have been isolated from the outbreaks, but A_5 has been the most prevalent cause of recent outbreaks. Due to the severity of the problem, Italy began semiannual FMD vaccinations in April 1987. Italy reported 71 outbreaks in March 1987, 15 in April, 6 in May, and 2 in June. The European Economic Community (EEC) has proposed an FMD vaccination program for Italy aimed at immunizing 4.5 million swine in 60 days. Historically, the EEC has not helped defray vaccination costs. However, this proposal calls for the EEC and Italy to equally share the vaccination costs.

On October 9, 1987, West Germany reported an outbreak of FMD, suspected to be type O, in cattle pastured 1 km from an FMD vaccine plant in Hanover, Lower Saxony. A quarantine area of 18 km radius was set up around the affected area, and 33 cattle and 18 swine were slaughtered as a result of the outbreak. West Germany had experienced no FMD outbreaks since 1984.

The world reference laboratory for FMD in Pirbright, England, reported types O and $Asia_1$ FMD, from Nepal, and SAT_2 from Zimbabwe. Pirbright also reported **swine vesicular disease (SVD)** virus from Hong Kong.

The Netherlands was declared free of **African swine fever (ASF)** by the U.S. Department of Agriculture (USDA) on August 13, 1987. However, pork products must be heat treated or cured before being imported into the United States from the Netherlands, because that country has not been recognized by the United States as being free of FMD, SVD, and hog cholera.

Italy reported two outbreaks of ASF in April and two outbreaks in May 1987.

England reported an outbreak of **hog cholera (HC)** in Hampshire on August 30, 1987. Only one farm with 2,000 animals was involved. The source of the new outbreak is unknown; however, the imposition of new rules by the European Economic Community (EEC) requiring England to accept salted and cured pork products from EEC member countries where HC exists has raised the question of a possible introduction from the European Continent. This is the first outbreak in England since April 1986. The most recent prior outbreak was confirmed on April 10, 1986, and the most recent prior case was diagnosed in late June 1986. A total of 1,531 pigs on 10 farms were affected during the 1986 outbreak. European countries that reported HC during 1987 were the Federal Republic of Germany, Austria, Belgium, France, Italy, Luxembourg,

Holland, and Yugoslavia.

The Netherlands reported their first case of **contagious equine metritis (CEM)** on July 22, 1987. The disease was confirmed in a mare at a breeding station in the Province of North Brabant. Another breeding station in the Province of Gelderland was also placed under quarantine pending further investigations. Only artificial insemination will be permitted for the 400-500 mares present at these two breeding stations.

Spain reported an outbreak of **African horse sickness (AHS)** in the Central Provinces of Madrid, Avila, and Toledo. Horses died of the disease in late July 1987, but AHS was not officially confirmed until September 9. Spanish officials estimated 160 to 300 equines died from the disease.

The outbreak was attributed to an importation of zebras from Namibia to a safari park in the Alicante area in southeastern Spain. The zebras were later transferred to another park near Madrid, where the current outbreaks have occurred. No outbreaks have been reported in the Alicante area.

The affected areas lie along the Alberche and Manzanares Rivers where insect vectors are prevalent. Spanish authorities halted equine movements throughout the country, closed all borders, and initiated an insect control program and an equine vaccination campaign in the affected Provinces. All unvaccinated animals showing serological titers for AHS were to be sacrificed. A total of 38,000 doses of live AHS virus vaccine, obtained from South Africa, were given to horses, mules, and donkeys by September 23.

It was hoped that no further AHS would be reported during the month of October since cold weather would inhibit insect vector activity. However, deaths of 40 to 60 vaccinated horses and donkeys during October have raised questions about vector activity and the effectiveness of the live vaccine. A type 4 AHS virus was originally identified as the virus affecting horses in Spain. However, deaths in vaccinated horses and donkeys have been attributed to type 2 AHS virus. The South African vaccine used in Spain contained types 1 through 7. The most recent prior outbreak of AHS in Spain was in 1966.

Greece reported an outbreak of **sheep pox (SP)** on the small Greek island at Lesvos, in the Aegean Sea. Lesvos is only 10 miles from the Turkish coast, where SP is endemic. The disease was diagnosed clinically on October 19, in four milking flocks. Fifty of a total of 500 exposed sheep had clinical signs of the disease by October 19. Seventeen died. The younger animals were most severely affected. Contaminated birds or air currents carrying contaminated dust from the coast of Turkey were thought to be the source of the outbreak. Actions against SP taken by Greek animal health authorities are: restriction of movements of flocks on the affected island, isolation of affected animals and flocks, restriction of exports of animals and animal products from the island, disinfection of infected premises, and sanitary

measures at harbors and airports. The most recent prior outbreak of SP in Greece was in 1976. (Dr. Percy Hawkes, 301-436-8285)

Viral Turkey Rhinotracheitis

In the late 1960's, turkey coryza became endemic in Canada. The disease then spread to the United States where it was called turkey rhinotracheitis (TRT). The causative agent, Alcaligenese fecalis, is now widespread in the United States. It primarily affects turkey poults 4 to 8 weeks of age. (Simmons, D. G. 1984. In: Poultry Diseases, 8th ed., pp. 251-256, Iowa State Univ. Press, Ames, Iowa)

A disease of turkeys with signs similar to TRT, but of unknown cause, occurred in South Africa. There was heavy mortality and rapid spread throughout the country. The disease became endemic and virtually wiped out the South African turkey industry. (Stuart, J. C. 1986. Field experiences in the UK with turkey rhinotracheitis. Ninth Technical Conference, Bristol, England) In 1977, a more virulent outbreak of a similar disease occurred in Israel and eventually spread to West Germany (1980), Holland (1981), France (1981), and Spain. These latter outbreaks varied in severity between the North American form of the disease and the South African form. All of the European countries experienced a similar type of disease. Efforts to find an etiological agent were not successful. A variety of bacterial agents were incriminated but none were wholly accepted as the causative agent, nor did any fulfill Koch's postulates. (Andral, B.; Louazis, C.; Trap, D.; Newman, J. A.; Bennejean, G.; and Gaumont, R., 1985. Avian Diseases 29: 26-34)

In July 1985, TRT occurred on the eastern coast of Great Britain. The means of spread from France to East Anglia, England, is unknown. The index case may have existed for 1 to 2 weeks before the disease spread to adjoining farms. The disease occurred sporadically for the next 3 to 4 weeks, affecting about 10 farms. It is presumed that this early spread of the disease resulted from the movement of contaminated feed trucks or personnel from farm to farm. Then, in late August, the disease exploded throughout England and eventually to Wales. This was especially disastrous because 2 to 3 million poults are started there in large flocks and, in mid-to-late August, are divided into small lots and placed on farms throughout England to provide fresh turkeys for the holiday season. The disease did not spread to Scotland, where strict import regulations were enforced.

Early cases of TRT were seen by private veterinarians in Great Britain. Government laboratories were quickly alerted to the disease. For the first time, important epidemiological information was obtained. The disease spread slowly between farms at first. The first sign noticed was sneezing ("snicking"). The appearance of a clear mucous ocular discharge was followed by a nasal discharge and respiratory signs. Submandibular edema was also seen. Morbidity often reached 100 percent within 48 hours, although a longer period was seen when buildings were divided into pens. The disease swept through entire turkey farms in 3 to 5 days. All ages of birds were affected. Mortality varied from 5 to 50 percent. The disease

appeared to be more severe during the late fall and winter months, affecting breeder hens, causing white egg shells, and severely decreasing egg production. This was in contrast to the TRT associated with A. fecalis infection, where breeder hens were not affected. Mortality was high in two flocks that were stressed by the onset of egg production. Postmortem lesions were few. Tracheitis, purulent nasal exudate, and submandibular edema were the most characteristic findings.

Several bacterial agents were isolated: Escherichia coli, A. fecalis, Moraxella anatipestifer, and Pasteurella spp. However, when the mucous exudate was passed through bacterial filters, the bacteria-free filtrate produced a mild form of the disease in 3- to 10-day-old poults, and a viral agent was reisolated. Viral isolation attempts were successful using specimens collected early in the course of the disease.

The virus grew in turkey embryos, causing stunting and death at varying intervals after inoculation. It could also be passed in chicken embryos with no apparent effect. Workers at the Houghton Research Station, England, were able to grow the virus in chicken kidney and fibroblast cells. French workers also isolated the virus and adapted it to monkey vero cell cultures. Workers at the Central Veterinary Laboratories in Weybridge, England, isolated the virus by using chicken and turkey embryos and incubating the embryonated eggs beyond the time usually considered necessary for virus isolation.

It has been difficult to prepare TRT virus in concentrations greater than 10^4 infective units per ml. Hemagglutination has not been shown with TRT virus. Results of electron microscopy and polyacrylamide gel electrophoresis indicate that the virus belongs to the Paramyxoviridae family. It is believed to be a member of the genus Pneumovirus and may be closely related to the mouse respiratory syncytial virus. The virus causes ciliostasis in the trachea of the embryo. Little else has been reported on its characteristics. (Cullen, A. 1987. Rhinotracheitis breakthrough. Poultry World, March issue)

First attempts to develop a TRT diagnostic test were hampered by the difficulty in growing the virus. The first successful test employed indirect immunofluorescence. Recently, an enzyme-linked immunosorbent assay (ELISA) test appeared to work satisfactorily. (Grant, M., Baxter-Jones, C., and Wilding, G. P. 1987. An enzyme-linked immunosorbent assay for the serodiagnosis of turkey rhinotracheitis infection. Veterinary Record. 120:279-280)

Early in the history of TRT there were reports of a new disease syndrome in broiler breeder chickens, called swollen head syndrome (SHS). With the development of the ELISA test, there is now serological evidence that TRT in turkeys and SHS in chickens may be caused by the same viral agent. When the TRT agent is inoculated into specific pathogen-free (SPF) chicks, it produces a mild respiratory disease. The virus has not been isolated from field cases of SHS. (Wyeth, P. J., 1987. Antibodies to TRT in chickens with swollen head syndrome. Veterinary Record. 120:286)

In May of 1986, Veterinary Services placed a ban on the importation of turkey hatching eggs from Great Britain because shipments were about to begin and very little information was available about TRT. Sera from six turkey flocks in six States were sent to Great Britain in October 1986 where they were found to be negative to tests for TRT. In February of 1987, a technician from Central Veterinary Laboratories traveled to the National Veterinary Services Laboratories (NVSL), Ames, Iowa, with necessary antigens and plates, and demonstrated the TRT ELISA procedure to the Diagnostic Virology Section. NVSL subsequently tested other turkey flocks in the United States for TRT with negative results.

Policy has been established to allow the importation of turkey eggs for hatching in the United States under restricted conditions. These conditions require sentinel birds in an isolated breeding flock to remain free of TRT. After hatching in the United States, an 8-week brooding period must be continued in isolation without evidence of TRT before the poults can be released.

At first, the movement of chicken hatching eggs was not banned because there was no proof that the TRT virus caused SHS. Upon receiving information that TRT virus may cause SHS, a policy was established stating that chicken hatching eggs, imported from countries other than Canada, must be certified to come from breeder flocks serologically negative for the TRT agent. All chicken hatching eggs imported since October 1, 1986, are being traced and tested for TRT. Twelve of 30 flocks traced up to this time have been tested and found negative.

The United States and Canada are considered to be free of viral TRT. The situation in Europe is being followed closely. Serum from suspected flocks will be submitted to NVSL for testing. Additional information will be provided when it becomes available. (Dr. Earl Grass, 301-436-8950)

Bongo Embryo Exchange

The U.S. Department of Agriculture (USDA) is cooperating with the Cincinnati Zoo in a program that may allow the exchange of bongo antelope embryos with Kenya. The zoo plans to collect and cryopreserve embryos from captive bongo herds at the Cincinnati and Los Angeles Zoos and the Mt. Kenya Game Ranch at Nanyuki, Kenya. The United States embryos will be transferred into common eland at the Nairobi Wildlife Park. Kenyan embryos will be transferred into surrogate eland and bongos at the Cincinnati Zoo. Kenyan veterinarians will participate in the collections, cryopreservations, and transfers. It is hoped that the Kenyan Government will be able to acquire funds for the application of the new technology to other native wildlife and thus help to increase the reproductive potential of rare species.

In January 1987, the Cincinnati Zoo applied to the Office of the President of Kenya for permission to conduct a bongo embryo research project in Kenya. In March, the USDA specified the conditions under which importation of Kenyan embryos may be permitted and submitted a draft statement to the national animal

health authorities in Kenya. If the USDA protocol is accepted and the zoo is permitted to carry out the research in Kenya, the program may begin early in 1988. (Dr. H. D. Harris, 301-436-8499)

Focus On--Swine Vesicular Disease

Swine vesicular disease (SVD) is a contagious viral infection of swine, clinically indistinguishable from foot-and-mouth disease (FMD), vesicular stomatitis (VS), and vesicular exanthema of swine (VES). It produces vesicular lesions and erosion of the epithelium of the mouth, tongue, snout, lips, nose, and feet. The morbidity is variable--up to 100 percent--but mortality is very low.

SVD Etiology

SVD virus is a single-stranded RNA virus, classified as a Picornavirus. Its diameter varies from 28 to 32 nm, density is 1.34 grams/ml and sedimentation coefficient is 150S. The virus is stable at 50°C in 1 molar magnesium chloride. It is inactivated by sodium hydroxide, potassium permanganate, and iodophores in 30 minutes at 4°C, and in less than 2 minutes at 25°C in 8 percent formaldehyde or 0.04 percent sodium hypochlorite.

SVD virus is serologically related to the human enterovirus coxsackievirus B-5 (CB-5) (Graves, J. H., 1973, Nature Lond., 245: 3142). Antisera to CB-5 neutralizes SVD virus and sera from pigs recovered from SVD infection neutralizes CB-5 virus. Also, there is 50 percent homology between the RNA's of SVD and CB-5 viruses. Due to the close relationship between SVD and CB-5 viruses, and serological evidence of SVD virus infection in laboratory workers, it is believed that this virus may have originated from coxsackie B5 virus. (Brown, F.; Wild, T. F.; Rowe, L. W.; Underwood, B. O.; and Harris, T. J. R., 1976. J. Gen. Virol., 31:231-237)

When Lai and co-workers inoculated CB-5 virus into pigs, clinical signs of disease were not observed and no viremia was detected, but the virus was isolated from nasal swabs and fecal samples. Viral antigen was not detected in tissues even though brain lesions were observed in inoculated and contact pigs. Antibody to CB-5 virus was detected in all inoculated and contact pigs (Lai, S. S.; McKercher, P. D.; and Moore, D. M., 1980. Comp. Immun. Microbiol. Infect. Dis., 2: 459-468)

SVD History

In 1966, a disease resembling FMD was first reported in Italian swine. The causative agent was identified as a porcine enterovirus. In 1970, during a trial of an experimental FMD vaccine in Hong Kong, vesicles were observed in swine on a farm where swine were not vaccinated. The disease spread to other swine farms. The isolated virus was serologically unrelated to the FMD virus. The third occurrence of the disease was in England. Based on clinical signs, the disease was again thought to be FMD. However, serological tests failed to identify the disease agent as FMD virus. Instead, the causative agent was identified as a porcine enterovirus serologically related to the

viruses responsible for the vesicular disease outbreaks in Italy and Hong Kong. Since 1970, outbreaks of SVD have been reported from England, Austria, Belgium, France, Poland, West Germany, Switzerland, Italy, Taiwan, and Japan.

SVD Disease Signs

Lameness may be the first sign of the disease, followed by the appearance of vesicles and temperatures of 104°F to 106°F (40°C to 41.1°C). The affected pigs are reluctant to move, and their appetites are drastically reduced.

The characteristics of SVD are vesicles on the coronary bands of the feet, interdigital spaces, and foot pads. Vesicles are less frequent on the snout, lips, tongue, and teats. Vesicles, which rupture easily, are not restricted to the junction of skin and hoof, but on occasion extend up the affected limb. Following vesicle rupture, a shallow area of granular tissue remains. Epithelial areas on the feet adjacent to a vesicle may detach and form raw ulcerous areas. It is rare for the complete hoof to be lost, as sometimes occurs in FMD.

Diarrhea and nervous signs, including agitation, pushing the snout against the wall or ground, circling movements, and paralysis, usually accompany clinical SVD. Experimental studies indicated that SVD-infected pigs may develop encephalomyelitis.

Disease signs are more severe in young pigs than in older ones; however, recovery is fairly rapid and mortality is negligible except in newborn pigs.

SVD Pathogenesis

The incubation period of SVD varies from 2 to 7 days. A 30-hour incubation period was observed when pigs were inoculated with SVD virus.

Pigs are readily infected by feeding them meat containing SVD virus. They develop vesicular lesions by the second day after experimental infection. Lesions first appear on the coronary band and then on the dewclaw, tongue, snout, lips, and foot pads. Viral antigen can be detected in the tonsil, dorsal epidermis and dermis of the tongue, epidermis of snout, and coronary band within 2 days after contact exposure, indicating that the virus first multiplies in epithelial cells. (Lai, S. S.; McKercher, P. D.; Moore, D. M.; and Gillespie, J. H., 1979. Am. J. Vet. Res., 40:463-468) Vesicles can also be produced by inoculating the virus intravenously, or intradermally in the feet.

The virus has been isolated from feces, blood, nasal discharge, and oesophageal-pharyngeal (OP) fluid of SVD virus-infected pigs and from nearly all tissues of fatal cases. Experimentally, the virus has been isolated from nasal swabs, OP fluid, and feces as early as 1 day after infection either by inoculation or by contact with infected pigs.

SVD Serology

Neutralizing antibody to SVD virus is usually detectable by the 7th day after infection and reaches peak concentration by the 28th day. The antibody then persists for about 60 days in recovered pigs. Recovered pigs resist the disease.

A report from Italy in 1982, cited by Donaldson and co-workers, suggested that SVD had become milder than the disease that was observed during the 1960's and early 1970's. (Donaldson, A. I., Ferris, N. P., Knowles, N. J., and Barnett, I. T. R., 1983. Res. Vet. Sci., 35: 295-300) A 1982 survey in Japan showed swine on 21 of 36 farms had neutralizing antibody to SVD virus even though they were healthy. In 1983, Donaldson and co-workers compared a 1972 SVD virus with four strains isolated during 1981-82 and found only minor differences in the results of immunodiffusion and cross-neutralization tests. However, in experimentally infected pigs, the 1972 isolate produced typical SVD lesions and a higher antibody titer than the four 1981-82 isolates. The 1981-82 isolates did not cause lesions in pigs but raised SVD antibodies 14 days after the infection.

Diagnosis

Field diagnosis: SVD in swine is clinically indistinguishable from FMD. All vesicular conditions in pigs should be considered as FMD unless there is sufficient evidence to establish a connection with a proven case of SVD. Diagnosis of SVD by clinical signs alone is not reliable.

Laboratory Diagnosis: Specimens for the diagnosis of either SVD or FMD should include vesicular fluid, the epithelial covering of vesicular lesions and tissues still attached to the edges of the lesions, unclotted blood, and serum.

SVD can be rapidly identified by the complement fixation (CF) test using vesicular epithelium as antigen and known reference antisera to all serotypes of SVD, VS, VES, and FMD. Results can be obtained within 3 to 4 hours of arrival of satisfactory specimens at the laboratory. The virus can also be identified in tissue cultures. The tissue cultures used are primary calf thyroid, primary calf kidney, and BHK-21, MVPK and IBRS-2 cell lines. SVD virus grows only in MVPK and IBRS-2 cell lines, thus differentiating it from other vesicular viruses. Cytopathic effects are observed after 24 to 48 hours. The virus is then identified by the CF test. Unlike FMD virus, SVD virus will not produce vesicular lesions when inoculated in the bovine tongue; however, it will produce lesions when inoculated into pigs. It will also cause paralysis in 1- to 6-day-old mice.

Serological tests routinely used for SVD diagnosis are counterimmunoelectrophoresis (CIEP), enzyme-linked immunosorbent assay (ELISA), serum neutralization (SN), and double immunodiffusion (DID).

Transmission

The spread of SVDV depends upon the movement of infected pigs and contaminated materials. The first appearance of SVD in Great Britain in 1972 is believed to have been a result of feeding pigs with improperly processed garbage containing imported SVDV-contaminated pork. In many instances, pigs were sent to slaughter before the disease was diagnosed in the affected herd. Many outbreaks in Great Britain from 1972 to 1981 were traced to transport vehicles contaminated by infected pigs.

Unlike FMD, SVD virus is not excreted by or exhaled from the

respiratory tract, but emanates primarily from ruptured vesicles. Airborne spread of SVD has not been reported and is not suggested by epizootiological data. Spread of disease on an affected farm may not occur in the absence of common open drainage or movement of infected pigs from one pen to another.

SVD Virus Survival

In the presence of organic matter, SVD virus withstands drying and freezing and can survive the fermentation and smoking processes used in the preparation of edible pork products. SVD virus has been known to survive 400 days in dried salami and pepperoni sausages and 780 days in intestinal casings (cited by Blackwell, J. H., 1984. J. Am. Vet. Med. Assoc., 184: 674-679). The virus also resists commonly used disinfectants and is stable at pH 2.5 to 12.0.

SVD Host Range

The range of susceptibility of various host species to the vesicular viruses is shown in the following table:

	Viruses			
Host Species	SVD	FMD	VES	VS
Horses	0	0	$\frac{+}{-}$	+
Cattle	0	+	$\frac{0}{-}$	+
Swine	+	+	+	+
Sheep and Goats	0	+	0	+
Man	$\frac{+}{-}$	$\frac{+}{-}$	0	+
Mice, 1-2 days old	$\frac{+}{-}$	$\frac{+}{-}$	0	+
Adult Mice	0	$\frac{+}{-}$	0	+
*Mink	$\frac{+}{-}$	$\frac{0}{-}$	0	0

0 = not susceptible $\frac{+}{-}$ = seldom infected + = susceptible

(*Sahu, S. P. and Dardiri, A. H., 1979. J. Wildlife Dis., 15:489-494).

SVD Control

The successful control of SVD in the United Kingdom was based on FMD control procedures (Loxam, J. G. and Hedger, R. S., 1983. Rev. Sci. Tech. Off. Int. Epiz., 2(1): 11-24). These procedures consisted of the following: (a) Slaughter and disposal of all infected and exposed pigs on a premises, (b) restriction of all movements onto and from affected premises, and (c) cleaning and disinfecting of contaminated premises. Premises were sprayed with a 1-percent solution of sodium hydroxide. All nonflammable surfaces were singed with a flame-gun 48 hours later. This procedure was repeated after 14 days. (d) Limited restocking was allowed 8 weeks after final cleaning. Sentinel pigs were placed in pens where infection was known to have been present. The sentinel pigs were inspected before arrival on the premises and at 3 weekly intervals thereafter. If no SVD signs appeared in the sentinel pigs, full restocking was allowed. (e) Waste food feeding was and is restricted. (f) Serological surveys are continuing. (Dr. S. P. Sahu, Foreign Animal Disease Diagnostic Laboratory, VS, APHIS, USDA, P.O. Box 848, Greenport, NY 11944)

FAD Report Editorial Committee

Editorial Committee membership has been changed. The current members are: Dr. E. I. Pilchard, Chairman; Dr. Gary Colgrove, Dr. Robert R. Ormiston, Dr. M. A. Mixson, Dr. M. J. Gilsdorf, Dr. Percy W. Hawkes, and Mrs. Betsy Nordin.

Subject Index

This subject index covers FAD Report volumes 10 through 15. It provides quick access to articles that contain information related to the index words. Subjects are cited by volume number, (issue number), page number or span of pages, and year of publication. Readers who desire to maintain a complete file of the indexed articles can obtain copies of prior issues by sending a request to the editor. A subject index will be published each year in the winter issue.

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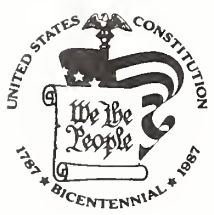

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